Overview of the most important mycotoxins for the pig and poultry husbandry

Overzicht van de meest belangrijke mycotoxines voor de varkens- en pluimveehouderij

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ABSTRACT

Mycotoxins are secondary metabolites produced by fungi, which may be present on a variety of crops. They are considered a major issue worldwide because of their harmful effects on animals. These contaminants lead to great economic losses, especially in pig and poultry husbandry. Over 400 mycotoxins have been identified. However, only few of them have a significant toxic effect and are of major concern. In this paper, the most important mycotoxins are described, including deoxynivalenol (DON), T-2 toxin (T-2), zearalenone (ZON), fumonisin B1 (FB1), ochratoxin A (OTA) and aflatoxin B1 (AFB1). For each toxin, its chemical structure, mode of action and symptoms of acute and chronic toxicity in pigs and poultry are discussed.

SAMENVATTING

Mycotoxines zijn secundaire metabolieten geproduceerd door verschillende schimmelsoorten die aanwezig kunnen zijn op diverse landbouwgewassen. Ze worden wereldwijd als een groot probleem aanzien omwille van hun schadelijke effecten op de humane en dierlijke gezondheid. De contaminatie leidt tot aanzienlijke economische schade, voornamelijk in de varkens- en pluimveehouderij. Er werden reeds meer dan 400 mycotoxines beschreven. Slechts enkele zijn echter belangrijk omwille van hun toxiciteit. In dit overzichtsartikel worden de belangrijkste mycotoxines beschreven, namelijk deoxynivalenol (DON), T-2 toxine (T-2), zearalenone (ZON), fumonisine B1 (FB1), ochratoxine A (OTA) en aflatoxine B1 (AFB1). Voor elk toxine worden de chemische structuur, het werkingsmechanisme en zowel de acute als chronische toxiciteit bij varkens en pluimvee weergegeven.

THE MYCOTOXIN ISSUE

Mycotoxins are secondary metabolites produced by fungi, mostly by saprophytic moulds growing on a variety of feed and foodstuffs (Turner et al., 2009). The name mycotoxin is a combination of the Greek word for fungus *'mykes'* and the Latin word *'toxicum'* meaning poison. Mycotoxin producing fungi can be divided in two classes, i.e. field and storage fungi. Field fungi, such as *Fusarium* species, produce mycotoxins during their growth in the field whereas storage fungi, such as *Aspergillus* and *Penicillium* species, produce mycotoxins after crop harvesting. Many factors may influence mycotoxin production, but temperature and humidity are commonly accepted as the most determining factors in the field, as well as during storage (Filtenborg et al., 1996). Mold contamination does not necessarily imply mycotoxin production. Furthermore, some mycotoxins are produced by only a limited number of fungal species, while others may be produced by a relative large range of genera. The prevalence of different fungal species is region dependent. *Fusarium* produced mycotoxins are more likely to occur in moderate regions, such as Western Europe and North America, whereas *Aspergillus* and *Penicillium* species are more prevalent in (sub)tropical regions. Nevertheless, ochratoxin A, produced by *P. verrucosum*, may also appear in more moderate regions (Duarte et al., 2010).

Contamination of feedstuffs with mycotoxins occurs worldwide at a higher level than generally assumed. In 2001, the Food and Agricultural Or-

ganization (FAO) stipulated that 25% of feedstuffs worldwide are contaminated (FAO, 2001). A more recent paper shows that one or more mycotoxins were detected in 82% of European crop samples (Monbaliu et al., 2010). In general, more than 50% of European samples are contaminated with deoxynivalenol at low levels (1-500 µg/kg), and 75-100% of the samples are contaminated with one or more mycotoxins (Streit et al., 2012).

To date, over 400 mycotoxins with toxic potential have already been described (Kabak et al., 2006). However, only few of them have distinct toxic effects. It was not until the 1960s that the first cases of mycotoxicosis were demonstrated. In Great Britain, more than 100 000 turkeys died due to liver necrosis and biliary hyperplasia (Turkey 'X' disease). The etiological agents were aflatoxins (Nesbitt et al., 1962). Since then, a tremendous amount of research has been conducted to investigate a variety of mycotoxins, their potential toxic effects and how to counteract their effects.

FUSARIUM MYCOTOXINS

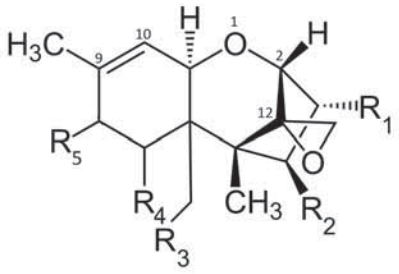
Fusarium fungi are field fungi, commonly occurring in Western Europe due to its moderate climate. They can produce a variety of mycotoxins. The most toxicologically important *Fusarium* mycotoxins are trichothecenes (including deoxynivalenol (DON) and T-2), zearalenone (ZON) and fumonisin B1 (FB1).

Although trichothecenes are generally produced by *Fusarium spp*., they may also be produced by other unrelated genera, such as *Stachybotrys, Trichoderma* and *Cephalosporium* (Ueno, 1985). Many trichothecene producing *Fusarium spp*. are causal agents of Fusarium Head Blight (FHB), root rot and foot rot in cereals. These include *F. graminearum, F. poae* and *F. culmorum* (Rocha et al., 2005). The most common ZON producing *Fusarium* fungi are *F. graminearum* and *F. culmorum*, but also *F. cerealis, F. crookwellense, F. semitectum* and *F. equisiti* (Zinedine et al., 2007). Fumonisins are produced by *F. verticillioides* (formerly *F. moniliforme*), *F. proliferatum* and other minor species including *F. nygamai* (Thiel et al., 1991).

Deoxynivalenol and T-2 toxin

Chemical structure

DON and T-2 are both trichothecenes. They are sesquiterpenoids, consisting of an alkene group at C-9-10, an epoxy at C-12-12, which is essential for its toxicity (Desjardins et al., 1993), and a variable number of acetoxy and hydroxyl groups. They have been classified into A, B, C and D toxins, depending on their functional groups (Ueno, 1977). Members of group A (e.g. T-2 and HT-2 toxin) do not contain carbonyl on C-8. Hydrolysis of ester groups leads to the formation of a basic trichothecene moiety with one to five hydroxyl groups. Group B (e.g. DON) differs from group A by the presence of a carbonyl group on C-8. Group C members (e.g. crotocine) have another epoxy group between the C-7 and C-8 or C-8 and C-9 positions. Compounds in group D, also called macrocyclic trichothecenes, (e.g. satratoxin G) include a macrocyclic ring between C-4 and C-15 (Wu et al., 2010) (Figure 1).



	T-2	HT-2	DON
R_1	-OH	-OH	-OH
R_2	-OAc	-OH	-H
R_3	-OAc	-OAc	-OH
R_4	-H	-H	-OH
R_5	$-OCOCHR_2CH(CHR_3)R_2$	$-OCOCHR_2CH(CHR_3)R_2$	=O

Figure 1. Chemical structure of T-2 toxin (T-2) , HT-2 toxin (HT-2) and deoxynivalenol (DON)

Mode of action

The most prominent molecular target of trichothecenes is the 60S ribosomal unit, where they prevent polypeptide chain initiation (T-2) or elongation-termination (DON) (Ueno, 1984). Thompson and Wannemacher (1986) demonstrated that T-2 is the most potent protein synthesis inhibitor, whereas DON is less potent. The addition of an acetyl chain (3a- or 15a-DON) further decreases its inhibitory potential. Furthermore, the de-epoxy metabolite of DON, de-epoydeoxynivalenol (DOM-1), has almost no inhibitory capacity. The main metabolite of T-2, namely hydrolyzed T-2 (HT-2), is also less potent than the parent compound. Trichothecenes also inhibit DNA and RNA synthesis, which is a secondary effect due to protein synthesis inhibition (Ueno, 1985). Furthermore, they inhibit mitosis, and cause loss of membrane function (Rocha et al., 2005). Finally, they activate mitogen-activated protein kinases (MAPKs), and induce apoptosis in a process called ribotoxic stress response (Pestka, 2007). As a consequence of MAPK activation, DON increases the expression and stability of cyclooxygenase-2 (COX-2) mRNA and hence the protein content in leukocytes, confirming its role in the inflammatory process (Moon and Pestka, 2002). Recently, it has been demonstrated that 15-acetyldeoxynivalenol (15a-DON) is a more potent activator of MAPK in vitro than DON or 3-acetyldeoxynivalenol (3a-DON) (Pinton et al., 2012). This is in contrast to what has generally been accepted.

Toxicity in pigs and poultry

The first symptoms of trichothecene intoxication were observed in the USSR in the 1930s where consumption of overwintered moldy feed resulted in massive outbreaks of alimentary toxic aleukia (ATA) in pigs. The symptoms included vomiting, diarrhea, leukopenia, hemorrhage, shock and death (Joffe and Palti, 1974).

Pigs are the most sensitive species to DON as well as to T-2, mainly due to their limited metabolic activity (Wu et al., 2010). High exposure of pigs to DON or 'vomitoxin' elicits abdominal distress, malaise, diarrhea, emesis and even shock or death (Pestka, 2010). The emetic effect is thought to be mediated through the affection of the serotonergic activity in the central nervous system or via peripheral action on serotonin receptors (SCF, 1999). T-2 is one of the most acute toxic mycotoxins. Acute mycotoxicosis in pigs is characterized by multiple hemorrhages on the serosa of the liver and along the intestinal tract (Weaver et al., 1978).

Although the LD_{50} values (the acute dose at which 50% of the tested animals die within 24 hours) are moderate, poultry are less susceptible to trichothecenes than pigs. The LD_{50} value is 5 mg/kg feed for T-2 and 140 mg/kg feed for DON (Chi et al., 1977). Acute intoxication of broiler chickens has several consequences including internal hemorrhage, mouth and skin lesions (necrohemorrhagic dermatitis), impaired feather quality and neural disturbances (Sokolovic et al., 2008).

Chronic exposure to lower doses of DON (≥ 50 µg/kg BW) and T-2 (≥ 10 µg/kg BW) induces growth retardation, weight gain suppression and feed refusal, mainly in pigs. The immune system is very sensitive to trichothecenes, and can be either stimulated or suppressed depending on the time, duration and dose of exposure (Pestka, 2008; Sokolovic et al., 2008). Low concentrations induce proinflammatory gene expression at mRNA and protein levels, while high concentrations promote leukocyte apoptosis. Trichothecenes, especially DON and T-2, can provoke reproductive and teratogenic effects, but exert no carcinogenic effect. The International Agency for Research on Cancer (IARC) has listed them as group 3 substance (non-carcinogenic) (IARC, 1993).

Zearalenone

Chemical structure

ZON is a resorcyclic acid, and has been given the trivial name zearalenone as a combination of *Giberella zeae* (now *Fusarium graminearum*), resorcyclic acid lactone, -ene (for the presence of the C-1,2 double bond) and -one (for the presence of C-6 ketone) (Urry et al., 1966) (Figure 2).

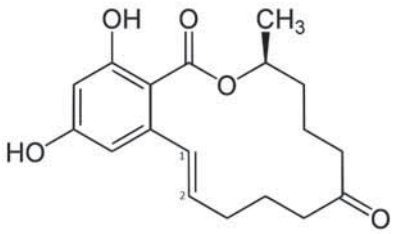


Figure 2. Chemical structure of zearalenone (ZON)

Mode of action

ZON can be listed as a non-steroidal or mycoestrogen (Tiemann and Dänicke, 2007). It resembles 17β-oestradiol, the principal hormone produced by the ovary, to allow ZON to bind estrogen receptors in target cells (Greenman et al., 1979). Estrogenic compounds diffuse in and out cells but are retained with high affinity and specificity by estrogen receptors. Once the estrogen receptor is bond, it undergoes a conformational change allowing the receptor to interact with chromatin and to modulate transcription of target genes (Kuiper et al., 1998). Not all compounds have the same affinity to estrogen receptors. It has been shown that the metabolites of ZON can express lower or even higher affinities to estrogen receptors than the parent compound. The metabolization of ZON occurs primarily in the liver, but a variety of organs show metabolization activity, such as intestine, kidney, ovary and testis, ZON is metabolized by 3α- and 3β-hydroxysteroid dehydrogenase (HSD) into α- and β-zearalenol (ZOL), respectively. β-ZOL has a 2.5 times lower affinity to the estrogen receptor, whereas α-ZOL has a 92 times higher binding affinity than ZON. The metabolization to β-ZOL can therefore be regarded as an inactivation pathway, whereas the metabolization to α-ZOL can be seen as an bioactivation pathway (Malekinejad et al., 2006). The rate of α- or β-ZOL production, and consequently the susceptibility, are species dependent. Pigs are the most sensitive species, which has been confirmed by in vitro data demonstrating that pig liver microsomes dominantly convert ZON into α-ZOL. Poultry and cattle metabolize ZON to a large extent into β-ZOL, confirming their relative resistance (Malekinejad et al., 2006; Zinedine et al., 2007).

Following or simultaneously with these hydroxylation reactions, phase II metabolization reactions take place. ZON and its metabolites are conjugated with glucuronic acid, catalyzed by uridine diphosphate glucuronyl transferases (UDPGT) (Olsen et al., 1981). Glucuronidation enhances the water solubility of compounds, thus enhancing renal elimination. On the other hand, it prolongs the total body residence time due to enterohepatic circulation, which has been demonstrated for ZON in pigs (Biehl et al., 1993).

Toxicity in pigs and poultry

The acute toxicity of ZON is rather low. The oral LD_{50} values in mice and rats vary from 4000 to >

20,000 mg/kg BW (Hidy et al., 1977). The specific manifestations of ZON in pigs are dependent on the dose, age, stage during estrus cycle and pregnancy or not. ZON intoxication leads to an estrogenic syndrome and affects primarily the reproductive tract and mammary gland. In young gilts, 1-5 mg/kg feed induces clinical signs, such as hyperemia, edematous swelling of the vulva and even vaginal or rectal prolaps (Minervini and Dell'Aquila, 2008). At lower doses (0.05 mg/kg feed), ZON induces vulva redness, swelling of the mammary gland and numerous vesicular follicles and some cystic follicles on the ovaries (Bauer et al., 1987). In cyclic animals, nymphomania, pseudopregnancy, ovarian atrophy and changes in the endometrium have been reported. During pregnancy, ZON can induce embryonic death, reduces embryonic survival, decreases fetal weight and induces teratogenic effects in piglets characterized by various genital abnormalities (D'Mello et al., 1999). In boars, ZON can suppress testosterone levels, testes weight and spermatogenesis, while inducing feminization and suppressing the libido (Zinedine et al., 2007).

ZON has little effect on poultry reproduction due to their well-developed metabolization pathways. Feeding mature chickens a diet contaminated with ZON up to 800 mg/kg does not have any effect on their reproductive performance (Allen et al., 1980; Allen et al., 1981a). Moreover, this contamination level does not have negative effects on the performance of mature broiler chickens nor of young turkey poults (Allen et al., 1981b). However, feeding 100 mg ZON/kg feed to mature female turkeys, reduces the egg production by 20% (Allen et al., 1983).

Next to their major effects on reproduction and the hormone system, ZON and its metabolites may also effect other organ systems. ZON has been shown to be hemotoxic. It disrupts the blood coagulation process, alters hematological parameters, such as hematocrit count, mean cell volume and number of platelets), as well as some serum biochemical parameters, such as aspartate aminotransferase, alanine aminotransferase, serum creatine and bilirubin (Maaroufi et al., 1996). ZON is also hepatotoxic, shown by altered serum biochemical parameters (Zinedine et al., 2007). Genotoxic and immunotoxic effects of ZON have also been demonstrated in vitro and in mice (JECFA, 2000). The IARC has classified ZON as a non-carcinogenic component (group 3) (IARC, 1993). Nevertheless, more recent data demonstrate that ZON stimulates the growth of mamma tumors containing estrogen receptors, indicating that it can play a role in tumor development after chronic exposure (Ahamed et al., 2001; Yu et al., 2005).

Fumonisin B1

Chemical structure

The chemical structure of fumonisins was first identified in 1988 (Gelderblom et al., 1988). To date, more than 28 fumonisin homologues have been identified. Fumonisin B1 is the most thoroughly investigated

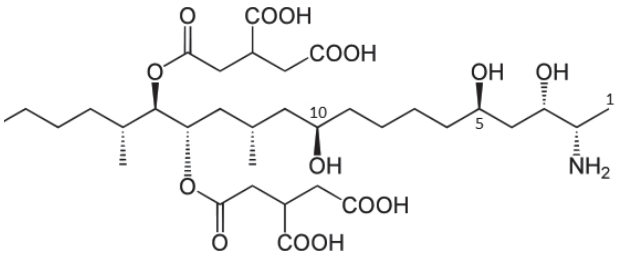


Figure 3. Chemical structure of fumonisin B1 (FB1)

because of its toxicological importance (Figure 3). Fumonisin B2, B3 and B4 are less prevalent, and differ structurally from FB1 in the number and placement of hydroxyl groups, i.e. a loss of a hydroxyl group on C-10, C-5 and both C-5 and C-10, respectively (Voss et al., 2007). The primary amine function is necessary for the toxicological activity of fumonisins. Deamination leads to a significant reduction in toxicity (Lemke et al., 2001). Cleavage of the tricarballylic acid side chains of FB1 leads to a less toxic metabolization product, named hydrolyzed fumonisin B1 (HFB1) (Grenier et al., 2012).

Mode of action

Fumonisins competitively inhibit sphinganine N-acyl transferase (ceramide synthase) and consequently disrupt the ceramide and sphingolipid metabolism (Merrill et al., 2001; Riley et al., 2001) (Figure 4). The inhibition of ceramide synthase consequently leads to an accumulation of free sphinganine (Sa), and to a lesser extent of sphingosine (So), and to a decrease of complex sphingolipids formation. The increase of free Sa leads to an increased Sa:So ratio in tissues and body fluids, which has been demonstrated to be a suitable biomarker for fumonisin exposure in mammals and avian species (Haschek et al., 2001). This increase is dose- and time-dependent, and is opted to occur rapidly and even at low levels (Voss et al., 2007). The increased concentrations of Sa and So, their phosphate adducts and a reduced ceramide concentration all contribute to the apoptotic, cytotoxic and growth inhibitory effects of fumonisins (Merrill et al., 2001). Moreover, the decrease of complex sphingolipids itself appears to contribute to the cellular effects of FB1 as well (Yoo et al., 1996) (Figure 4).

Toxicity in pigs and poultry

Signs of acute fumonisin intoxication include non-species specific symptoms, such as hepatotoxicity and renal failure, as well as species specific symptoms on target organs. The well-described pathology in horses is called equine leukoencephalomacia (ELEM), where the brain is targeted. In pigs, primarily, the heart tissue is affected, leading to cardiac insufficiency and consequently to pulmonary edema, called porcine pulmonary edema (PPE). FB1 as causal agent of PPE was first identified in 1992 (Osweiler et al.,

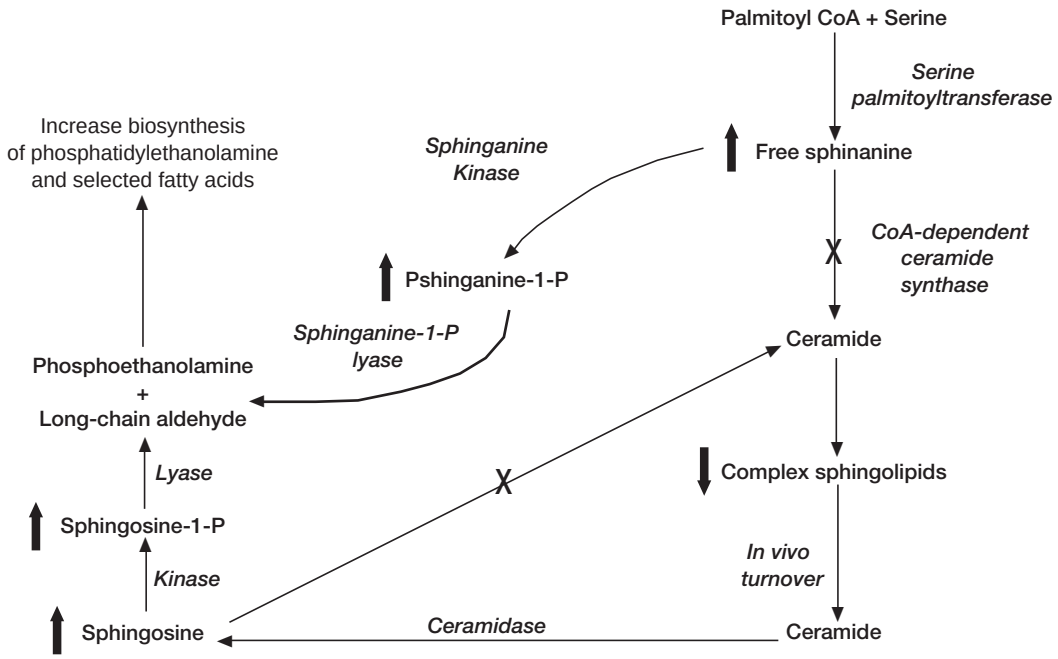


Figure 4. Mode of action of fumonisins (Voss et al., 2007).

1992). Thousands of pigs died in the USA due to the consumption of corn contaminated with *F. verticillioides*. Symptoms of this intoxication are reduced feed intake, followed by respiratory distress and cyanosis a couple of days later and finally death due to hydrothorax and acute pulmonary edema (Haschek et al., 2001). Poultry are quite resistant to fumonisin toxicity. Nevertheless, they may be at risk as well. In large areas in the world, the major part of their diet consists of maize, which can be highly contaminated (Diaz and Boermans, 1994). High doses (up to 300 mg/kg feed) are needed to induce clinical toxicity including decreased weight gain and liver failure in broiler chickens (Ledoux et al., 1992). In general, high doses are needed to induce toxicity as fumonisins have a very low oral bioavailability (Martinez-Larranaga et al., 1999). Turkeys are also quite resistant to fumonisin toxicity, although they are more susceptible than chickens (Weibking et al., 1994).

In mammals and poultry, immunosuppression has been demonstrated after chronic fumonisin exposure. This is economically important as adverse effects on the immune system may lead to increased pathogen susceptibility and lowered vaccinal response (Voss et al., 2007). Next to their effect on heart, liver and immune function, fumonisins exert reproductive, teratogenic and carcinogenic effects in laboratory animals (Howard et al., 2001; Riley et al., 2001; Voss et al., 1996a; Voss et al., 1996b). The IARC has classified FB1 as a group 2B compound (possibly carcinogenic to humans) (IARC, 1993).

ASPERGILLUS AND *PENICILLIUM* MYCOTOXINS

Aspergillus and *Penicillium* fungi occur worldwide, and are able to produce several mycotoxins. Toxicologically, the most important ones are OTA and aflatoxin B1 (AFB1). OTA was first isolated in 1965 from *A. ochraceus* (Van der Merwe et al., 1965). It is primarily produced during storage by *A. ochraceus* in tropical and warmer regions, and by *P. verrucosum* in more temperate regions (Duarte et al., 2010). AFB1 is mainly produced by strains of *A. flavus* and *A. parasiticus*, but also by other minor species, such as *A. nomius, A. bombycis* and *A. pseudotamari*, all occurring in tropical climates (Bennett and Klich, 2003).

Ochratoxin A

Chemical structure

OTA consists of a dihydroisocoumarin subunit, linked to phenylalanine by a peptide bond (Mally and Dekant, 2009) (Figure 5). Cleavage of the dipeptide bound induces the formation of ochratoxin alpha (OTα), a nontoxic metabolite. Other major but less toxic ochratoxins are ochratoxin B and C, which differ from OTA by the loss of the chlorine on C-5 or ethylester formation on the carboxyl function at C-11, respectively (Duarte et al., 2011; el Khoury and Atoui, 2010; Wu et al., 2011).

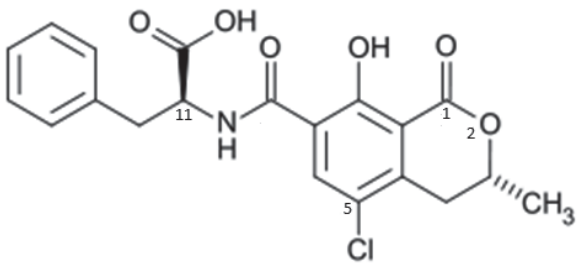


Figure 5. Chemical structure of ochratoxin A (OTA).

Mode of action

OTA does not act through a single well-defined mechanism, but it disturbs cellular physiology in multiple ways (Marin-Kuan et al., 2008). It seems that the primary effects are associated with the inhibition of the enzymes involved in the synthesis of the phenylalanine tRNA-complex, thus interfering with the phenylalanine metabolism. In addition, it stimulates lipid peroxidation (Bennett and Klich, 2003). It also disturbs the cellular mitochondrial respiration (Wei et al., 1985) as the open lactone moiety is structurally analogous to mitochondrial enzymes, including ATPase, succinate dehydrogenase and cytochrome C oxidase. OTA is also considered carcinogenic amongst laboratory animals (Group 2B compound) (IARC, 1993), although the mode of action has not been well described yet (Mally, 2012). The suggested molecular target is histone acetyltransferases (HATs). These enzymes are critical in the regulation of a diverse range of cellular processes, including gene expression, DNA damage repair and mitosis through posttranslational acetylation of histone and nonhistone proteins (Czakai et al., 2011; Mally, 2012).

Toxicity in pigs and poultry

Dietary human exposure to OTA has long been suspected to have been involved in Balkan endemic nephropathy (BEN), which occured in the 1950s. However, no direct proof can be put forward (Pfohl-Leszkowicz, 2009). The first report of OTA intoxication in animals was in the 1960s and 1970s in Denmark, where mycotoxic porcine nephropathy (MPN) has been correlated with OTA ingestion (Krogh et al., 1973). The kidneys are the main target organ of OTA.

Considerable species differences in sensitivity to acute OTA toxicity have been demonstrated (O'Brien and Dietrich, 2005). Pigs are particularly sensitive to OTA because of its long serum half-life and tissue accumulation. This is sustained by high protein affinity and enterohepatic and renal recirculation. Poultry species eliminate OTA faster than mammals, leading to a lower accumulation level. The half-life of OTA in pig plasma is 20-30 times longer than that in poultry plasma, leading to a higher OTA contamination level and incidence in pigs (Duarte et al., 2011). This difference is also demonstrated in the difference of the LD_{50} value in pigs and poultry: an oral LD_{50} value of 1 mg/kg BW for pigs versus 3.3 mg/kg BW for chickens and 5.9 mg/kg BW for turkeys (El-Sayed et al., 2009; Peckham et al., 1971).

Following chronic exposure to lower levels of OTA, the kidneys are again primarily affected, causing mycotoxic nephropathy in pigs as well as in chickens (Stoev et al., 2010). Several pathological changes may be observed, varying from desquamation and focal degeneration of tubular epithelium cells to peritubular fibrosis and thickening of the basal membrane (O'Brien and Dietrich, 2005). This leads to renal insufficiency, but not to tumor promotion in poultry and mammals species. In addition, OTA is hepatotoxic, teratogenic and immunotoxic (Duarte et al., 2011).

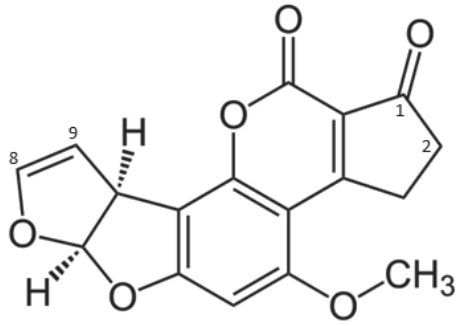


Figure 6. Chemical structure of aflatoxin B1 (AFB1).

Aflatoxin B1

Chemical structure

Over a dozen different aflatoxins have been described. Based on their fluorescence under UV-light (blue or green), the four major aflatoxins are called aflatoxin B1, B2, G1 and G2, of which AFB1 is the most toxic (Squire, 1981). The structure of AFB1 was first elucidated by Asao, et al. (1965), and is difurocoumaro-cyclopentenone (Figure 6). Other aflatoxins have different substitutions, but they all share the basic coumarine structure.

Mode of action

Aflatoxins are converted by cytochrome P450 enzymes (phase I metabolization) to the reactive 8,9-epoxide form, which is essential for the toxicity. The responsible converting enzymes in mammals are mainly CYP1A2 and CYP3A4 (Gallagher et al., 1996). In chickens and turkeys, the corresponding enzymes are CYP2A6 and to a lesser extent CYP1A1 orthologs (Diaz et al., 2010a, b). The epoxide metabolite can bind to both DNA (causing genotoxicity) and proteins (causing cytotoxicity). More specifically, it binds to guanine residues of nucleic acids (Doi et al., 2002). Moreover, aflatoxin B1-DNA adducts may result in guanine-cytosine (GC) to thymine-adenine (TA) transversions (Bennett and Klich, 2003). This leads to irreversible DNA damage, and causes hepatocellular carcinomas (Eaton and Gallagher, 1994).

The toxic epoxide metabolite may be detoxified by gluthatione conjugation (phase II metabolization) or hydrolysis by an epoxide hydrolase to AFB1-8,9-dihydrodiol (AFB1-dhd) or by metabolization to less toxic compounds such as aflatoxin M1 (AFM1) or Q1 (AFQ1) (Diaz et al., 2010b; Gallagher et al., 1996). This AFM1 for example is the main metabolite formed in cattle and is excreted in milk. As this metabolite still has carcinogenetic properties (10 times lower than

AFB1), maximum limits in milk for human consumption have been established (0.05 µg/kg) (European Commission, 2010).

Toxicity in pigs and poultry

The main biological effects of aflatoxins are carcinogenicity, immunosuppression, mutagenicity and teratogenicity (Ramos and Hernandez, 1997). Because of its pronounced carcinogenic effect, even in humans, the IARC has classified AFB1 as a group 1 compound (IARC, 1993).

Acute aflatoxicosis in pigs has been described (Coppock et al., 1989). The intake of contaminated feed (0.2 mg/kg) leads to reduced feed intake and body weight gain, impaired liver and immune functions and altered serum biochemical parameters (Harvey et al., 1990; Lindemann et al., 1993; Rustemeyer et al., 2010 and 2011).

Poultry species are the most susceptible food animals to AFB1. Feed contaminated with even small amounts of AFB1 results in significant adverse health effects, including death. On autopsy, generally, a firm and pale liver is found, the target organ of aflatoxins. When chickens are chronically exposed to lower doses, growth retardation occurs as well as immunological alterations and histological changes in the liver ('fatty liver') (Newberne and Butler, 1969). Turkeys are even more susceptible to aflatoxin intoxication than chickens, attributed to a combination of efficient AFB1 activation and deficient detoxification by phase II enzymes such as glutathione-S-transferase (Klein et al., 2000). Feeding a diet contaminated with 1 mg/kg AFB1 to turkeys resulted in 88% mortality rate (Kubena et al., 1991). Lower concentrations induce poor performance, decreased organ weights, liver damage and changes in biochemical serum values (Coulombe, 1993; Kubena et al., 1991).

CONCLUSION

In this review, several toxicologically important mycotoxins are described. This information may help the veterinary practicioner to better understand the mycotoxin issue and its implications. More than 400 mycotoxins have been identified; however, not all of them have been thoroughly investigated regarding their potential harmfull effects. For example, enniatins (ENNs) and beauvericin (BEA) are *Fusarium* mycotoxins commonly occuring in a variety of feedstuffs (Devreese et al., 2013). Nevertheless, little is known about their toxicokinetics and toxicodynamics in farm animals. Therefore, there is an urge to fully characterize other, lesser known mycotoxins.

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Uit het verleden

DE NACHTZWALUW: MELKDIEF EN MASTITISVERWEKKER

"De nachtzwaluw is een vogel die van God gemaakt is om bij nachte het venijn (schadelijk ongedierte) te pakken, dat in de lucht vliegt en om de beesten verkeert. Hij is onhoorbaar in zijn vlugge, heeft eenen bek die wijd opengaat tot onder zijne oogen. (…).

Dit is de waarheid, maar haddet gij over (voor) twee, drie duust jaar de geleerden en nu nog sommige lieden onder 't volk te rade geweest, zij hadden u gansch een andere historie van de nachtzwaluw uiteen gedaan. Een groot wijde bek, bij nachte vliegen, gezien geweest omtrent koeien of geetenuiers – om kwellend ongedierte te vangen – dat ongedierte, dat, ongevangen, ontstekingen veroorzaakt op de uierspenen, 't was genoeg: de nachtzwaluw melkt bij nachte de melkkoeien en trekt de geeten drooge, zeide men, en alle beesten die hij gemolken heeft, besmet hij den aan den uier.

Dat is onwaarheid, die bij alle natiën tot nog onlangs voor waarheid aangenomen werd en 't bewijs daarvan zit in de namen; aigitheles heet (noemt) hem Aristoteles, de groote wijzaard, en hij beschuldigt hem daarbij openlijk (van) melkdieverije; caprimulgus heet hij in 't Latijn, goatsucker in 't Engelsch, Milchsauger in 't Duitsch, tette-chèvre in 't Fransch. In 't Vlaamsch en kenne ik hem maar eenen name, die hem wonder wel past, en die hem van alle andere vogels onderscheiden houdt, te weten nachtzwalm of nachtzwaluwe."

Nvdr: ***Aegotheles*** is een geslacht van vogels uit de familie van de dwergnachtzwaluwen (*Aegothelidae*), voorkomend in Azië en Australië. Onze **nachtzwaluw** (***Caprimulgus europaeus***) behoort tot de nachtzwaluwenfamilie (*Caprimulgidae*), niet verwant met de zwaluwen of de gierzwaluwen. In het Nederlands kent men ook de benaming **geitenmelker**, een letterlijke vertaling van het Latijnse *Caprimulgus*.

Uit: Guido Gezelle's *Uitstap in de Warande* (1865-1870 en 1882, 6de uitgave 1927, De Meester, Wetteren, 1927).

L. Devriese (met dank aan M. Adriaen en P. Desmet)